

In the Claims:

Prior to examination, please amend the claims as follows:

1. (Original) A method for altering the load of a Hepatitis virus in a host organism infected with said virus, comprising the modulation of the complex formation of a heterogeneous nuclear ribonucleoprotein (hnRNP) K or a functional fragment thereof with a regulatory region on the Hepatitis virus genome.
2. (Original) The method of claim 1, wherein the said virus is selected from the group consisting of mouse Hepatitis virus, woodchuck Hepatitis virus, ground squirrel Hepatitis virus, arctic ground squirrel Hepatitis B virus, human Hepatitis B virus (HBV), duck Hepatitis B virus, heron Hepatitis B virus, sheld goose Hepatitis B virus, snow goose Hepatitis B virus, Ross' goose Hepatitis B virus, stork Hepatitis B virus, woolly monkey Hepatitis B virus, orangutan Hepadnavirus, GB virus B, and human Hepatitis C virus (HCV).
3. (Currently amended) The method of ~~claims 1 or 2~~ claim 1, wherein the host organism is a microorganism or a mammal.
4. (Currently amended) The method of ~~any of claims 1 to 3~~ claim 1, wherein the mammal is selected from the group consisting of a rat, a mouse, a squirrel, a hamster, a woodchuck, an orang-utan, a woolly monkey, a chimpanzee, a tamarin (*saguinus oedipus*), a marmoset and a human.
5. (Currently amended) The method of ~~any of claims 1 to 4~~ claim 1, wherein the modulation of said complex formation is achieved by means of altering the total amount of a variant of heterogeneous nuclear ribonucleoprotein (hnRNP) K or a functional fragment thereof in the cell.
6. (Currently amended) The method of ~~any of claims 1 to 5~~ claim 1, comprising administering a compound that modulates the complex formation of a hnRNP K protein

or a functional fragment thereof with the regulatory region on the Hepatitis virus genome.

7. (Currently amended) The method of ~~any of claims 1 to 6~~ claim 1, wherein the regulatory region is enhancer II of a hepadnavirus.
8. (Original) The method of claim 7, wherein the enhancer II region comprises positions 1554 to 1645 of the Hepatitis B virus genome.
9. (Currently amended) The method of ~~any of claims 1 to 8~~ claim 1, wherein the said virus is the human Hepatitis B virus.
10. (Currently amended) The method according to ~~any of claims 1 to 9~~ claim 1, where the method is an in-vivo method for the identification of suitable compounds that modulate said complex formation.
11. (Original) The method of claim 10, comprising administering a suitable compound for modulating the complex formation of a hnRNP K or a functional fragment thereof protein with the regulatory region on the Hepatitis virus genome.
12. (Original) The method of claim 11, further comprising measuring the number of Hepatitis virus particles in the host organism over a period of time.
13. (Original) The method of claim 11 or claim 12, further comprising:
comparing the obtained results with those of a control measurement.
14. (Original) The method of claim 13, wherein the control measurement comprises the use of a compound that does not modulate the complex formation of said hnRNP K protein or a functional fragment thereof with the regulatory region on the Hepatitis virus genome.
15. (Original) The method of claim 13, wherein the Hepatitis virus is the human Hepatitis B virus, the regulatory region is enhancer II, and wherein the control measurement

comprises the use of a variant of HBV that does not contain adenine at position 1752 of the virus sequence.

16. (Currently amended) The method of ~~any of claims 1 to 5 and 5-15~~ claim 1, wherein the host organism is a recombinant microorganism expressing a hnRNP K protein or a functional fragment thereof.
17. (Original) The method of claim 15, wherein the microorganism is a cell derived from liver tissue.
18. (Original) The method of claim 17, wherein the cell is of or derived from a hepatocellular or a hepatoblastoma cell line.
19. (Currently amended) The method of claim 18, wherein the cell line is selected from the group consisting of HepG2, Hep3B, HCCM, PLC/PRF/5, Sk-Hep-1, Snu182, HuH-6 **[[or]] and** HuH-7.
20. (Currently amended) A method of ~~any of claims 1 to 19~~ claim 1, wherein the complex formation of the hnRNP K protein or a functional fragment thereof with the said regulatory region of the Hepatitis virus is reduced by means of a nucleic acid molecule.
21. (Original) The method of claim 20, wherein the nucleic acid molecule is RNA or DNA.
22. (Currently amended) The method of claim 21, wherein the nucleic acid molecule is selected from the group consisting of an aptamer, a micro RNA (miRNA) molecule **[[or]] and** a small interfering RNA (si-RNA) molecule.
23. (Currently amended) The method of claim 22, wherein the nucleic acid ~~molecules~~ molecule is a si-RNA molecule comprising ~~the sequence~~ a sequence selected from the group consisting of SEQ ID NO: 2, SEQ ID NO: 4, SEQ ID NO: 6, SEQ ID NO: 8 **[[or]] and** SEQ ID NO: 10.

24. (Currently amended) A method of ~~any of claims 1 to 23~~ claim 1, wherein the interaction of a hnRNP K protein or a functional fragment thereof with a regulatory region of the Hepatitis virus is modulated by a compound that modulates the phosphorylation status of cellular components.
25. (Original) The method of claim 24, wherein the compound alters the degree of phosphorylation of a hnRNP K protein or a functional fragment thereof.
26. (Original) The method of claim 24, wherein the compound alters the intracellular quantity of hnRNP K proteins or functional fragments thereof.
27. (Currently amended) The method of ~~any of claims 24 to 26~~ claim 24, wherein the compound is an agonist or antagonist for a molecule on the cell surface.
28. (Original) The method of claim 27, wherein the molecule on the cell surface is a receptor.
29. (Currently amended) The method of claim 28, wherein the receptor is selected from the group consisting of a receptor tyrosine kinase, a membrane receptor with associated tyrosine kinase activity, [[or]] and a G protein coupled receptor.
30. (Currently amended) The method of claim 29, wherein the receptor is selected from the group consisting of a receptor for a platelet derived growth factor, a receptor for erythropoietin, a receptor for tumor necrosis factor, a receptor for leukaemia inhibitory factor, a receptor for an interferon, a receptor for insulin, a receptor for an insulin-like growth factor, a receptor for an interleukin, a receptor for a fibroblast growth factor, a receptor for a granulocyte-macrophage colony stimulating factor, a receptor for a transforming growth factor, [[or]] and a receptor for an epidermal growth-factor.
31. (Currently amended) The method of ~~any of claims 27 to 30~~ claim 27, wherein the agonist or antagonist is a protein.
32. (Currently amended) The method of claim 31, wherein the protein is selected from the group consisting of a mutein based on a polypeptide of the lipocalin family binding to

a receptor tyrosine kinase, a glubody binding to a receptor tyrosine kinase, an immunoglobulin binding to a receptor tyrosine kinase, ~~[[or]]~~ a protein based on the ankyrin scaffold binding to a receptor tyrosine kinase, and ~~[[or]]~~ crystalline scaffold~~[[,]]~~ binding to a receptor tyrosine kinase.

33. (Original) An in-vitro method of identifying a compound capable of altering the formation of a complex between a hnRNP K protein or a functional fragment thereof, and a Hepatitis virus or a functional fragment thereof that contains the enhancer II region, comprising contacting the components that form said complex with each other.
34. (Original) The method of claim 33, comprising:
 - (a) adding a compound to the test tube that modulates the complex formation of said hnRNP K protein or a functional fragment thereof with the enhancer II regulatory region on the Hepatitis virus genome, and
 - (b) detecting the said complex formation.
35. (Currently amended) The method of claim 34, wherein the detection is performed by a member selected from the group consisting of a suitable spectroscopic, photochemical, photometric, fluorometric, radiological, enzymatic or thermodynamic method, and a method ~~or is~~ based on cellular effects.
36. (Original) The method of claim 35, wherein the photochemical method comprises a cross-linking reaction.
37. (Original) The method of claim 35, wherein the spectroscopic method comprises the use of fluorescence correlation spectroscopy.
38. (Original) The method of claim 35, wherein the photometric detection method comprises the use of a label that is optically detectable.
39. (Original) The method of claim 35, wherein the radiological detection method comprises the use of a radioactive label.

40. (Currently amended) The ~~[[use]]~~ method of ~~claims 38 or 39~~ claim 38 that comprises the use of an electrophoretic mobility shift assay.
41. (Currently amended) The ~~[[use]]~~ method of ~~any of claims 38 to 40~~ claim 33 comprising the use of at least two nucleic acid molecules comprising the enhancer II region of the Hepatitis B virus DNA sequence, one of which does not contain adenine at position 1752 of the said sequence.
42. (Original) The method of claim 41, wherein the nucleic acid molecule not containing adenine at position 1752 is used for a control measurement.
43. (Currently amended) The ~~[[use]]~~ method of ~~any of claims 33 to 42~~ claim 33 for the in-vitro screening for potential compounds that are useful for treatment of Hepatitis infection due to their inhibition of the complex formation of a hnRNP K protein or a functional fragment thereof with a Hepatitis virus, comprising the simultaneous screening of compound libraries on multiple-well microplates using automated work stations.
44. (Original) The method of claim 43, wherein the Hepatitis infection is caused by HBV.
45. (Currently amended) ~~The use of A method for treating a Hepatitis infection comprising administering to a subject~~ a compound selected from the group consisting of aptamers, micro RNA molecules, small interfering RNA molecules, compounds that modulate the absolute quantity of hnRNK proteins in a cell, compounds that modulate the degree of phosphorylation of hnRNP K proteins, ~~[[and]]~~ agonists ~~or antagonists~~ for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase and antagonists for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase ~~for the manufacture of a medicament for the treatment of Hepatitis infection~~, wherein the viral load is altered via the modulation of the complex formation of a hnRNP K protein with a regulatory region on the Hepatitis virus genome.

46. (Currently amended) The ~~[[use]]~~ **method** of claim 45, wherein the agonist or antagonist for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase is **selected from the group consisting of** a mutein based on a polypeptide of the lipocalin family **binding to a receptor tyrosine kinase**, a glubody **binding to a receptor tyrosine kinase**, an immunoglobulin binding to a receptor tyrosine kinase, ~~[[or]]~~ a protein based on the ankyrin **scaffold binding to a receptor tyrosine kinase**, ~~[[or]]~~ a protein based on the crystalline scaffold~~[[,]]~~ binding to a receptor tyrosine kinase, a membrane receptor with associated tyrosine kinase activity, ~~[[or]]~~ **and** a G protein coupled receptor.
47. (Currently amended) ~~The use of~~ **A method for treating a Hepatitis infection comprising administering to a subject** a compound identified by a method of ~~any of claims 8 to 44~~ **claim 8**, wherein the viral load is altered via the modulation of the complex formation of a hnRNP K protein with a regulatory region on the Hepatitis virus genome.
48. (Currently amended) The ~~[[use]]~~ method of ~~any of claims 45 to 47~~ **claim 45**, wherein the Hepatitis infection is caused by HBV.
49. (Currently amended) ~~The use of~~ **A method of diagnosing a Hepatitis infection comprising using** a compound selected from the group consisting of aptamers, micro RNA molecules, small interfering RNA molecules, compounds that modulate the absolute quantity of hnRNK proteins in a cell, compounds that modulate the degree of phosphorylation of hnRNP K proteins, and agonists ~~or antagonists~~ for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase, antagonists for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase, **antagonists for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase, wherein using said compound the viral load is altered via the modulation of the complex formation of a hnRNP K protein with a regulatory region on the Hepatitis virus genome for the manufacture of a composition for the diagnosis of Hepatitis infection.**

50. (Currently amended) The ~~[[use]]~~ **method** of claim 49, wherein the agonist or antagonist for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase **is selected from the group consisting of** a mutein based on a polypeptide of the lipocalin family **binding to a receptor tyrosine kinase**, a glubody **binding to a receptor tyrosine kinase**, an immunoglobulin **binding to a receptor tyrosine kinase** ~~[[or]]~~, a protein based on the ankyrin **scaffold binding to a receptor tyrosine kinase** ~~[[or]]~~ a protein based on the crystalline scaffold~~[[,]]~~ binding to a receptor tyrosine kinase, a membrane receptor with associated tyrosine kinase activity, ~~[[or]]~~ **and** a G protein coupled receptor.
51. (Currently amended) ~~The use of~~ **A method of diagnosing a Hepatitis infection comprising using** a compound identified by a method of ~~any of claims 8 to 44~~ **claim 8, wherein using said compound the viral load is altered via the modulation of the complex formation of a hnRNP K protein with a regulatory region on the Hepatitis virus genome for the manufacture of a composition for the diagnosis of Hepatitis infection.**
52. (Currently amended) ~~The use of~~ **A method of evaluating a Hepatitis infection, wherein by using** at least two nucleic acid molecules comprising the enhancer II region of the Hepatitis B virus DNA sequence, one of which does not contain adenine at position 1752 of the said sequence **the viral load is altered via the modulation of the complex formation of a hnRNP K protein with a regulatory region on the Hepatitis virus genome, one of which does not contain adenine at position 1752 of the said sequence, for the manufacture of a kit or a composition for use in evaluating Hepatitis infection.**